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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

ALEXANDER KAMB
Junior Party
(Patent No. 6,090,578),

v.

DAVID H. BEACH, DOUGLAS J. DEMETRICK,
MANUEL SERRANO AND GREGORY J. HANNON,
Senior Party
(Application No. 09/016,869).

Patent Interference No. 105,459 (MPT).
(Technology Center 1600)

DECISION - Bd. R. 125

1
2
3 Before: **SALLY GARDNER LANE, MICHAEL P. TIERNEY, and**
4 **JAMES T. MOORE Administrative Patent Judges.**
5 **TIERNEY, Administrative Patent Judge.**

6
7 This interference is before a motions panel for a decision on preliminary
8 motions. This interference is decided on the briefs as neither party requested oral
9 argument.

1 I. Introduction

2 This interference is directed to immunoreactive antibodies, i.e.,
3 antibodies that are reactive with a particular set of antigens. The antigens
4 involved in this interference are mammalian MTS1 (multiple tumor
5 suppressor) polypeptides, where the term MTS1 polypeptide includes p16
6 polypeptides.

7 Kamb's '578 specification describes the multiple tumor suppressor
8 (MTS) gene as having a locus that contains at least two coding sequences,
9 MTS1 and MTS2. Kamb teaches that MTS1 includes the entire coding
10 sequence of p16 plus two introns. Kamb describes monoclonal antibodies
11 directed to MTS polypeptides as having use in assays as well as
12 pharmaceuticals.

13 Beach's involved '869 specification describes cell cycle regulatory
14 proteins ("CCR proteins") including p16 proteins. Beach describes making
15 monoclonal antibodies that are specifically reactive to p16 protein. Beach
16 states that anti-CCR antibodies can be used for a variety of purposes
17 including diagnostics and evaluating the levels of one or more CCR-proteins
18 in tissue or cells isolated from a bodily fluid.

19 There are six pending motions covering a wide range of issues
20 including anticipation, obviousness, written description, enablement, utility
21 and 35 U.S.C. § 120 benefit dates. The primary issue presented however, is
22 claim construction. More particularly, the parties dispute the meaning of
23 three key claim terms: immunoreactive, specifically immunoreactive and
24 mammalian MTS1 polypeptide. Generally, as discussed in the opinion
25 below, Beach has presented sufficient credible evidence of record to support
26 its proposed claim constructions whereas Kamb has not.

1 II. Findings of Fact

2 The record supports, by a preponderance of the evidence, the
3 following findings:

4

5 A. The Real Parties in Interest

6 1. Junior Party Kamb

7 1) The University of Utah Research Foundation is listed as the real party
8 in interest for Kamb U.S. Patent 6,090,578. (Kamb Designation of Real
9 Party in Interest, Paper 9).

10

11 2) Myriad Genetics is listed as the assignee on Kamb's '578 patent and
12 Myriad Genetics is listed on the correspondence address for the lead and
13 backup attorneys. (Kamb '578 and Kamb Designation of Lead and Backup
14 Counsel, Paper 8).

15

16 2. Senior Party Beach

17 3) Cold Spring Laboratory is the real party in interest for Beach, U.S.
18 Application 09/016,869. Beach Revised Identification of Real Party in
19 Interest, Paper 19).

20

21 4) Cold Spring Laboratory has licensed the interfering application to
22 MTM Laboratories and to Digene Corporation and states that the U.S.
23 Government may have certain rights to the invention due to federal grants
24 used by the inventors. (*Id.*).

1 B. Accorded Priority Benefit

2 1. Junior Party Kamb

3 5) Kamb is involved in the interference based upon Kamb's U.S. Patent
4 6,090,578, issued on July 18, 2000, which issued from U.S. Application
5 08/986,515, filed December 8, 1997. (Notice Declaring Interference, p. 3).

6

7 6) For the purpose of 35 U.S.C. § 102(g) priority, Junior Party Kamb has
8 been accorded the following priority benefit, i.e., earlier constructive
9 reduction to practice:

10 1) U.S. Application No. 08/480,810, filed June 7, 1995;
11 2) PCT/US95/03316, filed March 17, 1995; now U.S. Patent No.
12 5,801,938, issued September 1, 1998;
13 3) U.S. Application No. 08/251,938, filed June 1, 1994;
14 4) U.S. Application No. 08/215,087, filed March 18, 1994;
15 5) U.S. Application No. 08/215,086, filed March 18, 1994;
16 6) U.S. Application No. 08/227,369, filed April 14, 1994;
17 7) U.S. Application No. 08/214,582, filed March 18, 1994.

18 *(Id. at 5).*

19

20 2. Senior Party Beach

21 7) Beach is involved in the interference based upon Beach's U.S.
22 Application 09/016,869, filed January 30, 1998. (*Id. at 3*).

23

24 8) For the purpose of 35 U.S.C. § 102(g) priority, Senior Party Beach has
25 been accorded the following priority benefit, i.e., earlier constructive
26 reduction to practice:

27 1) U.S. Application No. 08/893,274, filed July 15, 1997, now U.S.

1 Patent No. 5,968,821, issued October 19, 1999;
2 2) U.S. Application No. 08/306,511, filed September 14, 1994,
3 now U.S. Patent No. 5,962,316, issued on October 5, 1999;
4 3) U.S. Application No. 08/248,812, filed May 25, 1994, now U.S.
5 Patent No. 5,889,169, issued March 30, 1999;
6 4) U.S. Application No. 08/227,371, filed April 14, 1994;
7 5) U.S. Application No. 08/154,915, filed November 18, 1993.
8 *(Id. at 5).*

9

10 C. The Count and Claim Correspondence
11 9) There is a single count in the interference, Count 1, which reads as
12 follows:

13 An antibody according to claim 2 of U.S. Patent No. 6,090,578 or
14 according to claim 104 of U.S. Application 09/016,869.

15
16 *(Id. at 4).*

17

18 10) Claim 2 of Kamb's involved '578 patent reads as follows:
19 An antibody immunoreactive with a mammalian MTS1 polypeptide
20 and not immunoreactive with other mammalian polypeptides.
21 (Kamb Clean Set of Claims, Paper 7).

22

23 11) Claim 104 of Beach's involved '869 application reads as follows:
24 An isolated antibody specifically immunoreactive with a p16 protein
25 comprising SEQ ID NO: 35.
26 (Beach Clean Set of Claims, Paper 5).

27

28 12) The claims of the parties are:

1 Kamb, U.S. Patent No. 6,090,578: 1-4
2 Beach, U.S. Application No. 09/016,869: 92, 93, 104 and 105
3 (*Id.*).

5 13) The claims of the parties which correspond to Count 1 are:

6 Kamb, U.S. Patent No. 6,090,578: 2 and 3

7 Beach, U.S. Application No. 09/016,869: 92, 93, 104 and 105

8 (*Id.*).

10 14) The claims of the parties which do not correspond to Count 1, and
11 therefore are not involved in the interference, are:

12 Kamb, U.S. Patent No. 6,090,578: 1 and 4

13 Beach, U.S. Application No. 09/016,869: None

14 (Id.).

15

D. Kamb's Involved Specification

17 15) Kamb's specification states that its invention relates to mutations in
18 the Multiple Tumor Suppressor (MTS) gene in human cancers and their use
19 in diagnosis and prognosis of human cancer. (KX 2008, Kamb '578, col. 1,
20 ll. 20-35).

21

22 16) Kamb's specification teaches that:

23 It is a discovery of the present invention that the MTS locus
24 (referred to in the prior art as Melanoma (MLM) locus), which
25 predisposes individuals to melanoma and other cancers, is a
26 gene encoding MTS1, which has been found to be an inhibitor
27 of Cdks, particularly Cdk4'. This gene is termed MTS1 herein.
28 It is also a discovery of the present invention that the MTS
29 locus contains a second coding sequence, termed MTS2, which

1 is very similar to MTS1 over part of its sequence. It is also a
2 discovery of the present invention that the MTS1 gene has two
3 separate promoters— α and β . When the α promoter is used the
4 resulting mRNA is composed of exon 1 α , exon 2 and exon 3.
5 This is referred to as MTS1. When the β promoter is used the
6 resulting mRNA is composed of exon 1 β , exon 2 and exon 3.
7 This is referred to as MTS1E1 β .

8
9 (Id. at col. 8, ll. 29-43).

10
11 17) Kamb's specification teaches that a review of a p16 mRNA sequence
12 revealed that MTS1 contained a stretch of 307 bp that was identical to a
13 portion of the p16 coding sequence. (Id. at col. 9, ll. 16-19).

14
15 18) Kamb's specification states that MTS1 was shown to include the
16 entire coding sequence of p16 plus two introns. (Id. at col. 9, ll. 20-22).

17
18 19) Kamb describes monoclonal antibodies directed to MTS polypeptides
19 as having use in assays as well as pharmaceuticals. (Id. at col. 14, ll. 38-57).

20
21 20) Kamb's specification states:

22 The term P16 is used interchangeably with MTS1 and MTS1
23 E1 β and is used to mean both MTS1 which encodes a p16 and
24 MTS1E1 β which encodes a p10. MTS1 and MTS1E1 β are two
25 forms of one gene, the two forms being dependent upon which
26 of two promoters is used for transcription. MTS2 is a separate
27 portion of the MTS region and it encodes a p15.

28
29 (Id. at col. 17, ll. 2-8).

30
31 21) Kamb's specification states that p16's biochemical function as a
32 potent inhibitor of a Cdk fits neatly with a model where MTS1 acts in vivo

1 as a general inhibitor of the onset of DNA replication. (*Id.* at col. 10, ll. 9-
2 11).

3

4 22) The terms "MTS Locus," "MTS gene," "MTS Nucleic Acids" and
5 "MTS Polynucleotide" are defined by Kamb as referring to polynucleotides
6 in the MTS region and are likely to be expressed in normal tissue. (*Id.* at
7 col. 16, ll. 30-33).

8

9 23) Kamb states that the defined MTS terms, "when applied to a nucleic
10 acid, refer to a nucleic acid which encodes a MTS polypeptide (including
11 p16), fragment, homolog or variant, including, e.g., protein fusions or
12 deletions." (*Id.* at col. 16, ll. 53-56).

13

14 24) Kamb's specification also specifically states that:

15 "MTS protein" or "MTS polypeptide" refer to a protein or
16 polypeptide encoded by the MTS locus (including MTS1
17 polypeptide, MTS2 polypeptide and MTS1E1 β polypeptide),
18 variants or fragments thereof. The term "polypeptide" refers to
19 a polymer of amino acids and its equivalent and does not refer
20 to a specific length of the product; thus, peptides, oligopeptides
21 and proteins are included within the definition of a polypeptide.
22 This term also does not refer to, or exclude modifications of the
23 polypeptide, for example, glycosylations, acetylations,
24 phosphorylations, and the like. *Included within the definition*
25 *are, for example, polypeptides containing one or more*
26 *analogs of an amino acid (including, for example, unnatural*
27 *amino acids, etc.), polypeptides with substituted linkages as*
28 *well as other modifications known in the art, both naturally*
29 *[sic] and non-naturally occurring. Ordinarily, such*
30 *polypeptides will be at least about 50% homologous to the*
31 *native MTS sequence, preferably in excess of about 90%, and*
32 *more preferably at least about 95% homologous.* Also
33 included are proteins encoded by DNA which hybridize under

1 high or low stringency conditions, to MTS-encoding nucleic
2 acids and closely related polypeptides or proteins retrieved by
3 antisera to the MTS protein(s).

4
5 (*Id.* at col. 18, ll. 28-50, emphasis added).

6

7 25) Kamb's specification states that an "amino acid sequence of an MTS
8 polypeptide (MTS1) is shown in SEQ ID NO:2." (*Id.* at col. 16, ll. 60-63).

9

10 26) Kamb's specification does not expressly define the term
11 "immunoreactive."

12

13 27) Kamb's specification does not expressly define the term "mammalian
14 MTS1 polypeptide."

15

16 E. Beach's Involved U.S. Application 09/016,869 (BX 1022)

17 28) Beach's involved '869 application is directed to cell-cycle regulatory
18 proteins ("CCR proteins") having an apparent molecular weight of 16 kDa.
19 (BX 1022, p. 4, Summary of the Invention).

20

21 29) Beach Figure 6 is said to depict a sequence alignment of a highly
22 conserved portion of p16, p15 and p13.5. (*Id.* at p. 10).

23

24 30) The '869 application states that an embodiment of the invention is
25 directed to antibodies that are specifically reactive with CCR proteins. (*Id.*
26 at p. 33, 1st full paragraph).

1 31) The '869 application teaches that anti-p16 monoclonal antibodies can
2 be made using standard methods. (*Id.*).

3

4 32) Beach '869 teaches that anti-CCR antibodies can be used for a variety
5 of purposes including diagnostics and evaluating the levels of one or more
6 CCR-proteins in tissue or cells isolated from a bodily fluid. (*Id.* at p. 34, 2nd
7 full paragraph).

8

9 33) Beach '869 describes immunoprecipitates that showed weak cross-
10 reactivity between p15 and p15.5 and p16 antiserum. (*Id.* at p. 55, 3rd full
11 paragraph).

12

13 34) Beach '869 claims 35 U.S.C. § 120 benefit of a series of applications
14 including U.S. Application 08/248,812, filed May 25, 1994, now U.S. Patent
15 5,869,169 and U.S. Application 08/154,915, filed November 8, 1993. (*Id.* at
16 p. 1, Related Applications).

17

18 2. Beach U.S. Pat. 5,869,169

19 35) Beach '169, like the '869 application, is directed to CCR proteins
20 having an apparent molecular weight of 16 kDa. ('169 patent, BX 1007,
21 Abstract).

22

23 36) The '169 patent SEQ ID NO:2 is said to be a p16 protein. (*Id.* at col.
24 4, ll. 62-65).

1 37) Like the '869 application, the '169 patent teaches that an aspect of the
2 invention is antibodies that are specifically reactive with a CCR protein. (*Id.*
3 at col. 20, ll. 46-47).

4

5 38) The '869 application states that anti-p16 monoclonal antibodies may
6 be made by conventional methods. (*Id.* at col. 20, ll. 48-51).

7

8 3. Beach, U.S. Application 08/154,915

9 39) Beach's '915 application is directed to methods and diagnostic kits for
10 diagnosing transformation of a cell. ('915 Application, BX 1008, Abstract).

11

12 40) The '915 application states that its method is particularly directed to
13 interaction of cyclins, PCNA, CDKs and low molecular weight
14 polypeptides, such as p16. (*Id.*).

15

16 41) The '915 application provides a sequence, SEQ ID NO:4, for
17 p16INK4, which is also known as p16 and INK4. (*Id.* at p. 24, ll. 23-28).

18

19 42) The '915 application describes raising antibodies to a p16INK4 fusion
20 protein. (*Id.* at p. 26, ll. 24-36).

21

22 43) The '915 application states that antibodies specifically reactive with
23 compounds of the quaternary complexes can be produced and that
24 monoclonal antibodies can be produced using known techniques. (*Id.* at p.
25 27, ll. 11-19).

1 44) The '915 application states that “[a]ntibodies specifically reactive
2 with CDK5, CDK4 and other CDKs in general, p21, p19, **p16** and cyclins
3 can also be produced using known methods.” (*Id.* at p. 27 ll. 19-20,
4 emphasis added).

5

6 45) The '915 application states that an embodiment of its invention is a kit
7 for detecting p16 levels in cells using antibodies specific for p16. (*Id.* at p.
8 28, ll. 14-17).

9

10 46) The '915 application also describes using labeled anti-p16 antibodies
11 to detect the presence of p16 in a sample of cells. (*Id.* at p. 29, ll. 21-22).

12

13 F. Person of Ordinary Skill in the Art

14 47) The art to which this interference pertains is antibodies that are
15 immunoreactive to a protein.

16

17 48) A person of ordinary skill in the art generally would have a Ph.D. in
18 biology or biochemistry and several years of post-graduate training and/or
19 research experience working with antibodies. (See, e.g., Krainer Dec., BX
20 1004 and Lanchbury Dec., KX 2049).

21

22 G. Declaration Testimony

23 The following facts present relevant highlights from Beach and
24 Kamb's testifying experts.

25

26 *Beach Declarations*

27 1. Dr. Adrian Krainer's Testimony

1 49) Dr. Krainer received a Ph.D. in biochemistry from Columbia
2 University in 1986. (BX 1004, ¶ 2).

3

4 50) Dr. Krainer is the scientific head of the Cold Spring Harbor
5 Laboratory Cancer Center Antibody Shared Resource, which routinely
6 generates mouse monoclonal antibodies. (*Id.* at ¶ 5).

7

8 51) Dr. Krainer has held numerous academic and editorial board
9 appointments and has been an author in over one hundred peer-reviewed
10 publications in the area of molecular biology. (*Id.* at ¶¶ 3-4).

11

12 52) Dr. Krainer testifies that a sequence for human p16 was described in
13 Serano et al., *Nature* 1993, 366 (6456): 704-7, which was submitted into the
14 interference record as BX 1003. (*Id.* at ¶ 10).

15

16 53) Dr. Krainer testifies that the 148 residues in Serano's p16 sequence
17 are identical to Beach SEQ ID NO:35 and are identical to residues 9-156 of
18 Kamb SEQ ID NO:2, with the exception that the Kamb sequence has a
19 glycine residue at position 35 whereas Serano has a valine residue at
20 corresponding position 27. (*Id.* at ¶¶ 10-13).

21

22 54) Dr. Krainer testifies that, while Beach's '169 patent does not specify a
23 particular method for calculating percent homology between two sequences,
24 one of ordinary skill in the art would understand that Serano's p16 is at least
25 94 % homologous with that of Kamb's SEQ ID NO:2. (*Id.* at ¶¶ 18-19).

1 55) Dr. Krainer testifies that the term “MTS1 polypeptide” as used in
2 Kamb’s ‘578 patent encompasses Serano’s 94% homologous human p16
3 polypeptide. (*Id.* at ¶ 20).

4

5 56) Dr. Krainer testifies that Kamb claim 2, which requires an antibody
6 that is “immunoreactive with a mammalian MTS1 polypeptide and not
7 immunoreactive with other mammalian polypeptides” encompasses an
8 antibody that specifically immunoreacts with a mammalian p16 polypeptide.
9 (*Id.* at ¶ 23).

10

11 57) Dr. Krainer testifies that Serano describes an antibody that specifically
12 immunoreacts with human p16/MTS1 polypeptide. (*Id.* at ¶ 32).

13

14 58) Dr. Krainer testifies that, as of March 1994, the generation of
15 monoclonal antibodies to an immunogenic antigen was a routine procedure
16 as demonstrated by Goding (1986), “Production of Monoclonal Antibodies,”
17 submitted into the interference record as BX 1005. (*Id.* at ¶ 34).

18

19 59) Dr. Krainer testifies that monoclonal antibodies were desired by
20 researchers as monoclonal antibodies were known in the art to possess
21 numerous advantages. (*Id.* at ¶ 35).

22

23 60) Dr. Krainer cites Gooding as teaching that specificity, degree of cross-
24 reaction, affinity and physical properties of a monoclonal antibodies may be
25 selected to suit individual needs. (*Id.*).

1 61) Dr. Krainer testifies that one of ordinary skill in the art would have
2 had a high expectation of success of generating monoclonal antibodies that
3 were specifically immunoreactive with mammalian MTS1 and not
4 immunoreactive with other mammalian polypeptides. (*Id.* at ¶ 38).

5

6 62) Dr. Krainer also testifies that Beach's '169 patent, which is allegedly
7 § 102(e) prior art to Kamb, clearly discloses monoclonal antibodies
8 specifically immunoreactive with p16 and, given Beach's teachings, testifies
9 that even an ordinary graduate student could have produced antibodies
10 specifically immunoreactive with p16 polypeptides. (*Id.* at ¶ 47).

11

12 63) Dr. Krainer prepared a second declaration that was submitted into the
13 record as BX 1037.

14

15 64) Dr. Krainer's second declaration provides testimony that, among other
16 things, demonstrates that Kamb's "mammalian MTS1 polypeptide" is not
17 limited to Kamb SEQ ID. NO: 2. In particular, Dr. Krainer demonstrates
18 that human MTS1, which is Kamb SEQ ID. NO:2, differs from other
19 mammalian MTS1 polypeptides such as horse and pig. (BX 1037, ¶¶ 11-
20 12).

21

22 *Kamb Declarations*

23 Kamb's preliminary motion did not rely upon expert testimony to
24 support its contention that Beach's specification fails to provide sufficient
25 written description and/or enablement for Beach's involved claims. Further,
26 Kamb's oppositions did not rely upon any Kamb expert testimony. Kamb

1 Reply 1 however, relies upon two declarations by Dr. Jerry Lanchbury, KX
2 2049 and KX 2050, which are summarized below.

3 1. Dr. Jerry Lanchbury's Testimony

4 65) Dr. Lanchbury received his Ph.D. in population genetics from the
5 University of Newcastle upon Tyne, UK. (KX 2049, ¶ 2).

6

7 66) Dr. Lanchbury has been employed at various immunogenics
8 companies since 1988. (*Id.*, ¶ 3).

9

10 67) Dr. Lanchbury is presently an Executive Vice President at Myriad
11 Genetics, Inc., which is the assignee listed on Kamb's involved patent. Dr.
12 Lanchbury has been has been employed at Myriad since 2002. (*Id.*, ¶ 1).

13

14 68) Dr. Lanchbury's first declaration (KX 2049) takes the position that
15 Beach's specification fails to provide a specific definition for the term
16 “specifically immunoreactive.” (*Id.*, ¶ 22).

17

18 69) Dr. Lanchbury testifies that Kamb's specification suggests that the
19 term “specifically reactive” antibodies are antibodies that react with a single
20 CCR-protein of interest, and include distinct antibodies that are individually
21 specifically immunoreactive with either p16, p15 or p13.5. (*Id.*, ¶ 23).

22

23 70) Dr. Lanchbury testifies that:

24 ... the antibodies in possession by the inventors, as described in
25 Beach's '869 Application were NOT antibodies that are “specifically
26 immunoreactive” with p16, since they were known to cross-react with
27 at least two other human proteins.

1 (Id., ¶ 33).

2

3 71) Dr. Lanchbury's second declaration (KX 2050) takes the position that,
4 if Beach's construction of the term "specifically immunoreactive" is correct,
5 then Beach's claims are anticipated by prior art. (KX 2050, ¶ 12).

6

7 72) Dr. Lanchbury's second declaration discusses several prior art
8 references and goes on to provide the following conclusions:

9 40. Antibodies to a specific protein can cross-react with other
10 proteins, even if the proteins have little or no amino acid
11 sequence identity, as long as noted in Dr. Krainer's declaration.
12 See Ex. 2021, pages 6-8. Other examples of antibody cross-
13 reactivity are in the literature including, Fadeel et al.,
14 *International Immunology*, 1998, 10(2):131-140, which teaches
15 that a monoclonal antibody to Fas/APO-1 is cross reactive with
16 multiple peptides that share no amino acid identity, but have
17 three dimensional homology, See Ex. 2038.

18

19 41. The scientific evidence indicates that the ARDs of ARD
20 containing proteins have significant primary amino acid
21 sequence identity. The amino acid identity and similarity
22 between the p16 ARDs and the ARDs of Ankyrin, suggests that
23 antibodies prepared against human Ankyrin, or other forms of
24 Ankyrin, can cross-react with similar epitopes present in p16.

25

26 42. The scientific evidence indicates that the ARDs of ARD
27 containing proteins have significant three-dimensional
28 structural homology. The three-dimensional structural
29 homology between the p16 ARDs and the ARDs of Ankyrin,
30 suggests that antibodies prepared against human erythrocyte
31 Ankyrin, or other forms of Ankyrin, can cross-react with
32 similar epitopes present in p16.

33

34 43. Based on these facts, I believe that antibodies that are
35 "specifically immunoreactive" with p16, as interpreted in Beach

1 Opposition 1, and Dr. Krainer's Second Declaration, were in the
2 art more than one year prior to Beach's earliest priority date.
3

4 (KX 2050, ¶¶ 40-43, emphasis in original).

5

6 III. Opinion

7 There are six motions awaiting decision, with Beach filing five
8 substantive motions and Kamb filing one. Generally, Beach filed three
9 motions alleging that Kamb's claims are unpatentable over prior art
10 (BM1,BM2 and BM4, Papers 26, 27 and 29), and a motion alleging Kamb's
11 claims lack utility (BM 3, Paper 28). Kamb filed a single motion alleging
12 that Beach's involved claims are unpatentable under 35 U.S.C. 112, 1st
13 paragraph, as they lack sufficient written description and/or enablement
14 (KM 1, Paper 22). Beach's remaining motion is a responsive motion that,
15 contingent upon the grant-in-part of Kamb's motion, requests that the Count
16 be redefined (BM 5, Paper 35).

17 The interference rules provide that:

18 To be sufficient, a motion must provide a showing, supported
19 with appropriate evidence, such that, if unrebutted, it would
20 justify the relief sought. The burden of proof is on the movant.

21 37 C.F.R. § 41.208(b). For the motions before us, the burden of proof is by
22 a preponderance of the evidence. The burden of showing something by a
23 preponderance of the evidence simply requires the trier of fact to believe that
24 the existence of a fact is more probable than its nonexistence before the trier
25 of fact may find in favor of the party who carries the burden. *Concrete Pipe*
26 & *Products of California, Inc. v. Construction Laborers Pension Trust for*
27 *Southern California*, 508 U.S. 602, 622, 113 S. Ct. 2264, 2279 (1993). Yet,
28 in rendering its factual findings:

1 . . . it is impermissible for the Board to base its factual findings
2 on its expertise, rather than on evidence in the record, although
3 the Board's expertise appropriately plays a role in interpreting
4 record evidence.

5
6 *Brand v. Miller*, 487 F.3d 862, 869 (Fed. Cir. 2007).

7 We begin our review of the parties' substantive motions with Kamb
8 Motion 1, as it raises a threshold issue, written description.
9 37 C.F.R. § 41.201.

10

11 A. Kamb Substantive Motion 1

12 Kamb Substantive Motion 1 requests that the Board enter judgment
13 that all of Beach's involved claims, claims 92, 93, 104 and 105, are
14 unpatentable to Beach for lack of written description and/or enablement
15 under 35 U.S.C. § 112, first paragraph. (KM 1, Paper 22, p. 2). In
16 particular, Kamb alleges that Beach's specification fails to provide sufficient
17 written description and/or enablement for "specifically immunoreactive"
18 antibodies, such as recited in Beach's involved claims. (*Id.*).

19

20 i. Beach's Involved Claims

21 All of Beach's involved claims are directed to isolated antibodies that
22 are "specifically immunoreactive." (Beach Clean Set of Claims, Paper 5).
23 Beach has two independent claims, claims 92 and 104. Beach's claim 92
24 requires the antibody to be immunoreactive with a 16 kD protein that
25 coprecipitates with CDK4 from cell lysates of SV40-transformed WI38 cells
26 in the presence of an anti-CDK4 antibody. (*Id.*). Beach claim 104 requires
27 the antibody be immunoreactive with a p16 protein defined by Beach SEQ
28 ID NO: 35. (*Id.*).

1

2 ii. Case Law on Written Description and Enablement

3 While the specifics of the cases concerning adequate written
4 description vary, the cases agree that the inquiry is factual and must be
5 assessed on a case-by-case basis. Moreover, because of the fact-sensitive
6 nature of the written description inquiry, the Federal Circuit has advised
7 against misapplication of precedent in this area. *See, Union Oil Co. of*
8 *California v. Atlantic Richfield Co.*, 208 F.3d 989, 1000 (Fed. Cir. 2000).

9 The purpose of the written description requirement is to ensure that
10 the inventor had possession, as of the filing date of the application relied on,
11 of the specific subject matter later claimed by the inventor. *Vas-Cath Inc. v.*
12 *Mahurkar*, 935 F.2d at 1563 (Fed. Cir. 1991). The inventor can
13 demonstrate possession by such descriptive means as words, structures,
14 figures, diagrams, formulas, etc., that fully set forth the claimed invention.
15 The inventor, however, needs to show that the inventor was "in possession"
16 of the invention by describing the invention, with all its claimed limitations,
17 not that which makes it obvious. *Lockwood v. American Airlines, Inc.*, 107
18 F.3d 1565, 1571-72 (Fed. Cir. 1997).

19 To comply with the enablement requirements of 35 U.S.C. 112, first
20 paragraph, a specification must adequately teach how to make and how to
21 use a claimed invention throughout its scope, without undue
22 experimentation. *Plant Genetic Systems N.V. v. DeKalb Genetics Corp.*, 315
23 F.3d 1335, 1339 (Fed. Cir. 2003). The scope of enablement is that which is
24 disclosed in the specification plus the scope of what would be known to one
25 of ordinary skill in the art without undue experimentation. *National*
26 *Recovery Technologies, Inc. v. Magnetic Separation Systems, Inc.*, 166 F.3d
27 1190, 1196, (Fed. Cir. 1999).

iii. Kamb Fails to Establish that Beach's Specification Lacks Sufficient Written Description and/or Enablement for "Specifically Immunoreactive" Antibodies

5 Kamb states that Beach’s involved ‘869 specification fails to
6 explicitly define the term “specifically immunoreactive.” (Paper 22, p. 2).
7 According to Kamb, the term “specifically immunoreactive” would
8 ordinarily be assumed to mean that the claimed antibodies are
9 immunoreactive with the recited proteins but not others. (*Id.* at 3). Kamb
10 goes on to argue that Beach’s specification describes antibodies that are
11 weakly cross-reactive with p16 serum, and thus, are not “specifically
12 immunoreactive” with p16. (*Id.* at 3, ll. 9-20).

13 Kamb has failed to demonstrate with credible evidence that its
14 proposed claim construction is correct. In essence Kamb requests that the
15 Board adopt a narrow claim construction, based upon an assumed meaning,
16 and then find that Beach's specification fails to support the narrowly
17 construed claim. Kamb's request violates the requirement that we provide
18 claims with their broadest reasonable interpretation in light of the
19 specification.

20 Beach's specification describes the use of anti-p16 antibodies for
21 diagnostic assays. (Beach '392 published application, ¶ 119).¹ Kamb

¹ Kamb Motion 1 cites, but does not identify, the exhibit number for the published application version of Beach's involved specification. (See, e.g., Paper 22, p. 3, ll. 9-20). Kamb's exhibit list identifies the published application as KX 2005, however, KX 2005 is not a published application and instead appears to be the as-filed Beach involved application. In reviewing Kamb's arguments we have relied upon the published application as) i) Beach's published application is a readily available public document, ii) no substantive difference is apparent between the published application

1 directs the Board's attention to Beach's co-immunoprecipitation of an anti-
2 p16 antibody with p15, p15.5 as well as p16 and Beach's statement that this
3 precipitation suggests that p15 and p15.5 were weakly cross-reactive with
4 the p16 antiserum. (Paper 22, p. 3, ll. 17-20, citing Beach published
5 application ¶ 189). Kamb states that the fact that p16 antiserum cross-reacts
6 with p15 and p15.5 is not surprising as p15 and p16 are highly similar. (*Id.*
7 at p. 3, ll. 21-28). Kamb goes on to argue that Beach's specification fails to
8 provide a single embodiment excluding the cross-reactivity and that, due to
9 the high degree of similarity between p16 and p15, it would be "highly
10 unpredictable" for a skilled artisan to make an anti-p16 antibody lacking
11 cross-reactivity with p15. (*Id.* at p. 3, line 29 to p. 4, line 5). Kamb also
12 argues that Beach fails to describe methods of making antibodies that can
13 distinguish proteins differing by one out of 148 amino acids and that it is
14 unpredictable as to whether such an antibody can even be made. (*Id.* at p. 4,
15 line 31 to p. 5, line 2).

16 Given the teachings in Beach's specification, it was Kamb's burden to
17 demonstrate that one of ordinary skill in the art would understand that
18 weakly cross-reactive antibodies were excluded from the claim. For, as
19 recognized by the Federal Circuit, it is rarely correct to interpret a claim as
20 excluding a preferred (and indeed only) embodiment and that such an
21 interpretation would require highly persuasive evidentiary support. *See, e.g.*,
22 *Rhodia Chimie v. PPG Industries Inc.* 402 F.3d 1371, 1377 (Fed. Cir.
23 2005); *Vitronics Corp. v. Conceptronic, Inc.*, 90 F.3d 1576, 1583 (Fed. Cir.
24 1996). Kamb's motion, however fails to provide such evidentiary support.

and the as-filed application, and iii) as our reliance does not result in
prejudice to Beach.

1 Kamb Motion 1 states that Beach's specification does not contain a
2 definition of the term "specifically immunoreactive." (Paper 22, p. 2, ll. 30-
3 32). Kamb states that its proposed claim construction is the one that "would
4 be assumed" by a person of ordinary skill in the art. (*Id.* at p. 3, ll. 1-3).
5 Kamb's motion however, fails to direct our attention to credible evidence of
6 record to support its assumed meaning. More particularly, Kamb's motion
7 contains a list of exhibits that identifies only a single exhibit as relied upon
8 to support the motion, exhibit 2001, which is a comparison of Beach's SEQ
9 ID NO:35 with wild-type p16. (Paper 22, Appdx. 1, List of Exhibits).
10 Additionally, while not identified in its exhibit list, Kamb cites Beach's
11 specification, more particularly, the published Beach application
12 2002/0082392A1, published June 27, 2002. Neither Beach's published
13 application nor the sequence comparison represent credible evidence that
14 Beach disavowed the anti-p16 antibody embodiment described in its
15 specification.

16 Kamb's lack of evidentiary support for its assumed claim
17 interpretation appears to be an attempt to have the Board rely upon its
18 expertise in place of evidence of record. Yet, as stated above, the Board
19 must base its underlying factual findings on the evidence of record and not
20 solely on its expertise. Accordingly, we will not assume that one of ordinary
21 skill in the art would interpret the disputed phrase in a manner inconsistent
22 with the general teachings of a specification absent credible and persuasive
23 evidence of record.

24 Additionally, Kamb's proposed claim construction is contrary to
25 providing claims with their broadest reasonable interpretation. Specifically,
26 Beach is an applicant, and as such, its claims are given their broadest
27 reasonable construction before issuance because the claims may be amended

1 to the proper scope and because it serves the public interest by reducing the
2 possibility that the claims will be construed more broadly after issuance than
3 they were during examination. 37 CFR § 41.200(b); *cf.*, *In re Bigio*, 381
4 F.3d 1320, 1324 (Fed. Cir. 2004). Indeed, the United States Patent &
5 Trademark Office is tasked with interpreting claims as broadly as their terms
6 reasonably allow. *In re Zletz*, 893 F.2d 319, 321 (Fed. Cir. 1989). *Zletz* held
7 that the Board erred in reading unwritten limitations into claims on appeal
8 and stated that it was incorrect for the Board to construe claims narrowly,
9 such as done in courts confronting issues of infringement and validity. Here,
10 Kamb's proposed construction would interpret Beach's claim as excluding
11 specific embodiments, i.e., narrowly construe Beach's claims. For such a
12 construction to be correct, Kamb must at a minimum explain why it is
13 unreasonable to adopt a broader construction that would encompass Beach's
14 described anti-p16 embodiment.

15 Based upon the evidence presented in Kamb Motion 1, we hold that
16 Kamb has failed to provide credible and sufficient evidence to demonstrate
17 that Beach's claimed "specifically immunoreactive" terminology excludes
18 the anti-p16 embodiment described in Beach's specification.

19 Kamb's arguments regarding Beach's alleged lack of written
20 description and enablement for Beach's involved claims require that the
21 Board first adopt Kamb's proposed claim construction for the disputed
22 terminology. As the Board does not adopt Kamb's proposed claim
23 construction, Kamb Motion 1 has failed to present a *prima facie* case that
24 Kamb is entitled to the relief it requests, i.e., holding Beach claims 92, 93,
25 104 and 105 unpatentable under 35 U.S.C. § 112, 1st paragraph.
26 Accordingly we **deny** Kamb Motion 1. We do not reach the issues and
27 evidence presented in Beach Opposition 1 or Kamb Reply 1 as we have

1 denied Kamb Motion 1 for failing to make out a *prima facie* case and the
2 Standing Order prohibits a party from presenting new issues or evidence in a
3 reply that were necessary to make a *prima facie* case for the relief requested
4 in the motion. (Standing Order “SO”, Paper 2 at ¶ 122.5 and
5 37 C.F.R. § 41.122(b) “All arguments for the relief requested in a motion
6 must be made in the motion. A reply may only respond to arguments raised
7 in the corresponding motion.”).

8

iv. New Issues and Evidence in Kamb Reply 1

10 Kamb alleges that its reply properly raises issues and evidence that
11 must be considered in the context of this interference.² We find however,
12 that Kamb Reply 1 (Paper 56) contains new issues and evidence in
13 contravention of the Standing Order (Paper 2). Specifically, the Standing
14 Order states that a reply that raises new issues or belatedly presents evidence
15 will not be considered. (SO at ¶ 122.5). The Standing Order identifies
16 examples of new issues and evidence as including evidence that is necessary
17 to make out a *prima facie* case for the relief requested and evidence that
18 could have been included with the motion. (*Id.*). *C.f., Kaufman Company,*
19 *Inc. v. Lantech, Inc.*, 807 F.2d. 970, 973 n. * (Fed. Cir. 1986); *McBride v.*
20 *Merrell Dow and Pharmaceuticals, Inc.*, 800 F.2d 1208, 1210-11 (D.C. Cir.
21 1986)(“We generally will not entertain arguments omitted from an
22 appellant’s opening brief and raised initially in his reply brief

² For example, Kamb Reply 1 states that:

All issues in Kamb Motion 1, Kamb Reply 1 and Kamb Opposition to Beach Responsive Motion, have been fairly raised and “fully developed.”

(Paper 56, p. 1, ll. 7-8).

1 Considering an argument advanced for the first time in a reply brief, then, is
2 not only unfair to an appellee, . . . but also entails the risk of an improvident
3 or ill-advised opinion on the legal issues tendered.”)(citations omitted).

4 While the exhibit list of Kamb Motion 1 identified only one (1)
5 exhibit as relied upon, the exhibit list for Kamb Reply 1 identifies fifty-two
6 (52) exhibits as relied upon. (Paper 22 and 54, Exhibit Lists). Furthermore,
7 Kamb Reply 1 provides nine (9) pages of argument as compared to Kamb
8 Motion 1, which provides four and a half (4 ½) pages. New issues raised in
9 Kamb Reply 1 include arguments that Beach's claims are unpatentable over
10 prior art and incorporation of arguments that were presented in Kamb's
11 opposition to Beach's responsive motion but not in Kamb Motion 1.
12 Examples of new evidence in Kamb Reply 1 include a citation to American
13 Heritage Dictionary and two declarations from Kamb's expert, Dr.
14 Lanchbury. Kamb however, alleges that these new exhibits were properly
15 submitted in connection with the Kamb reply. Kamb's allegations as to the
16 timeliness of the new evidence and the issues they present are discussed
17 below.

18

a. American Heritage Dictionary Citation

20 Kamb Reply 1 directs our attention to a lay dictionary, American
21 Heritage Dictionary, for a definition of the term “specific.” (Paper 56, p. 3,
22 citing KX 2042). Kamb states that the dictionary definition of specific
23 demonstrates that “Kamb’s interpretation of ‘specifically immunoreactive’
24 did not rest solely on attorney argument.” (*Id.* at p. 3, ll. 8-9). Kamb alleges
25 that its citation to the dictionary is proper as it does not seek to cure an
26 actual defect in Kamb Motion 1 and demonstrates that Kamb Motion 1
27 presented a *prima facie* case. (*Id.* at p. 3, ll. 22-25).

1 Kamb fails to explain why the citation to American Heritage
2 Dictionary could not have been presented in its motion as opposed to the
3 reply. (SO at ¶ 122.5, prohibits “new evidence that could have been
4 included with the motion”). The dictionary evidence raises new issues that
5 should have been addressed in the underlying motion, e.g., whether the
6 dictionary definition is in accord with the teachings of Beach’s specification,
7 does the cited definition accurately reflect the understanding of one of
8 ordinary skill in the immunological arts, etc. We hold that Kamb’s citation
9 to the American Heritage Dictionary definition of “specific” and the issues
10 raised thereby will not be considered as the evidence violates the prohibition
11 against evidence that could have been include have been included with the
12 motion.

13

14 b. First Declaration of Dr. Lanchbury

15 Kamb alleges that the first declaration of Dr. Lanchbury was properly
16 included in Kamb's reply. (Paper 56, p. 9). According to Kamb, the first
17 declaration of Dr. Lanchbury was submitted to expose the inconsistencies
18 that are present in Beach's expert declarations of Dr. Krainer. (*Id.* at p.9, ll.
19 1-4). Kamb states that the first declaration of Dr. Lanchbury was not
20 required to make out a *prima facie* case and that it was necessitated by the
21 "incredible construction" presented in Beach Opposition 1. (*Id.* at p. 9, ll. 4-
22 7).³

³ Kamb Reply 1 cites SO ¶ 121.5 as the basis for presenting the Lanchbury evidence in Kamb's reply. (Paper 56, p. 9, l. 7). Section 121.5 of the Standing Order concerns appendices. We believe that Kamb intended to cite section 122.5 of the Standing Order, which pertains to new issues in replies.

1 Kamb has failed to demonstrate that Beach’s proposed claim
2 construction was “incredible” and unforeseeable such that Dr. Lanchbury’s
3 declaration could not have been submitted in connection with the underlying
4 motion. Further, Kamb Motion 1 was denied for failing to make out a prima
5 facie case. As such, we did not consider Beach’s opposition or the
6 testimony of Dr. Krainer in reaching our decision for this motion.
7 Accordingly, we need not discuss Dr. Lanchbury’s testimony regarding Dr.
8 Krainer’s alleged inconsistencies and Beach’s proposed claim construction.

10 c. Second Declaration of Dr. Lanchbury

11 Dr. Lanchbury's second declaration asserts that Beach's claims, if
12 broadly construed, would be "inherently anticipated" by prior art. (Paper 56,
13 p. 7, ll. 29-31). Kamb Reply 1 states that Dr. Lanchbury's second
14 declaration should be admitted "because it is necessary to fully respond to
15 the incredible claim construction put forth in Beach Opposition 1." (*Id.*, p.
16 9, ll. 8-9).

17 We find Dr. Lanchbury's second declaration to be belatedly submitted
18 for the reasons discussed above with respect to Dr. Lanchbury's first
19 declaration. Further, even if we had reached the issues presented in Dr.
20 Lanchbury's second declaration, we would not have found Beach's claims
21 inherently anticipated by the art identified by Dr. Lanchbury. Inherency
22 requires inevitability and may not be established by probabilities or
23 possibilities. *In re Oelrich*, 666 F.2d 578, 581 (CCPA 1981); *Dreyfus v.*
24 *Sternau*, 357 F.2d 411, 415 (CCPA 1966). Dr. Lanchbury's testimony
25 however, merely concluded that the prior art *suggests* that known antibodies
26 could be "specifically immunoreactive" with p16. (KX 2050, ¶¶ 40-43).

d. The Standing Order Precludes Submission of Replies Raising New Issues and/or Belatedly Presented Evidence

5 The Standing Order clearly notifies a moving party that it is
6 impermissible to present new evidence and issues in a reply that were
7 necessary to make out a *prima facie* case. The Standing Order states that:

8 A reply that raises a new issue or belatedly presents evidence
9 will not be considered and may be returned. The Board will not
10 attempt to sort proper from improper portions of the reply.

11 (SO at ¶ 122.5). The Board finds that Kamb Reply 1 raised new issues and
12 belatedly presented evidence. Thus, in addition to not reaching the
13 opposition and reply due to Kamb Motion 1's failure to make out a prima
14 facie case, the Board does not consider Kamb's reply as it failed to comply
15 with the Standing Order's prohibition on new issues and belated presentation
16 of evidence.

B. Beach Responsive Motion 5 to Substitute a Count

20 Beach Responsive Motion 5 seeks to redefine the count in terms of
21 Kamb claim 2 or Beach claim 92. Consideration of Beach's responsive
22 motion is contingent upon the granting in part of Kamb Motion 1. (Paper
23 35, p. 1, ll. 5-7). (*Id.*). The contingency specified in Beach Responsive
24 Motion 5 did not come to pass. Accordingly, Beach Responsive Motion 5 is
25 dismissed as *moot*.

1 C. Beach Motion 2 for Judgment Based on Prior Art

2 Beach Motion 2 requests that Kamb's involved claims, claims 2 and
3 3, be held unpatentable over prior art. (Paper 27, p. 1). Specifically, Beach
4 alleges that Beach's 5,889,169 patent represents prior art to Kamb under
5 35 U.S.C. §102(e) and that the '169 patent anticipates Kamb claims 2 and 3.
6 (*Id.* at p. 3, line 19 to p. 4, line 5).⁴

7 Kamb claim 2 requires an antibody that is immunoreactive with a
8 mammalian MTS1 polypeptide and not immunoreactive with other
9 mammalian polypeptides. (Kamb Clean Set of Claims, Paper 7). Kamb
10 claim 3 depends from claim 2 and requires that the antibody be a
11 monoclonal antibody. (*Id.*).

12 The parties agree that Beach's '915 application discloses a human p16
13 amino acid sequence that is identical to residues 9-156 of Kamb's human
14 MTS1 polypeptide, with the exception that Kamb has glycine at position 35
15 whereas Beach has a valine residue at the corresponding position. (Paper
16 47, admitting facts 10-11). The parties disagree as to: 1) whether a human
17 p16 polypeptide is a "mammalian MTS1 polypeptide" as required by
18 Kamb's claims and; 2) whether Beach's '169 patent and '915 application
19 provide an enabling disclosure for "specifically immunoreactive" antibodies.
20 These issues are discussed in detail below.

21

22

⁴ Beach's '169 patent claims 35 U.S.C. §120 benefit of Beach's U.S. 08/154,915 application filed November 18, 1993, which precedes Kamb's earliest priority date of March 18, 1994. Kamb does not dispute the availability of the '169 patent as prior art under 35 U.S.C. § 102(e) or seek to antedate the '169 patent.

- 1 1. Claim Construction
- 2 a. Mammalian MTS1 Polypeptide
- 3 i. Mammalian MTS1 Polypeptide is Broader
- 4 than Kamb SEQ. ID. NO:2

5 Kamb contends that the term “mammalian MTS1 polypeptide” is
6 defined by Kamb’s specification as limited to Kamb SEQ ID NO:2. (Paper
7 47, p. 3, ll. 18-19). According to Kamb, its mammalian MTS1 polypeptide
8 “unequivocally lacks fragments and variants.” (*Id.* at p. 3, ll. 19-20). Kamb
9 concludes that Beach’s ‘169 patent and ‘915 application do not disclose
10 Kamb’s MTS1 polypeptide as Beach’s p16 differs from Kamb’s MTS1
11 polypeptide. (*Id.* at ll. 22-31).

12 Kamb’s proposed claim construction rests on its citation to the
13 following passage in its specification:

14 The coding sequence for an MTS polypeptide (MTS1) is shown
15 in SEQ ID NO:1, and the amino acid sequence of an MTS
16 polypeptide (MTS1) is shown in SEQ ID NO:2.

17 (KX 2008, col. 16, ll. 60-64). Kamb’s interpretation of this passage is based
18 upon attorney argument and is not supported by evidence from the vantage
19 point of one of ordinary skill in the art.⁵ For the reasons provided below, the
20 cited passage from Kamb’s specification is not enough to demonstrate that
21 Kamb’s mammalian MTS1 polypeptide is limited to the human MTS1
22 polypeptide sequence shown in SEQ ID NO. 2.

⁵ Kamb Opposition 2 contains an Appendix I (List of Exhibits Relied Upon in this Opposition). The Appendix lists only three (3) exhibits: Beach’s ‘915 application (KX 2002), Beach’s ‘392 publication and Kamb’s ‘578 patent (KX 2008).

1 Kamb’s proposed claim construction vis-à-vis prior art runs contrary
2 to its proposed construction vis-à-vis written description. Specifically,
3 Kamb alleges that for purposes of written description its “mammalian MTS1
4 polypeptide” encompasses variations but for purposes of prior art its
5 mammalian MTS1 polypeptide is limited to SEQ ID NO. 2 and does not
6 include variations. (See, e.g., Paper 41, p. 3).

7 Kamb Opposition 4 states that Kamb’s “mammalian MTS1
8 polypeptide” is fully characterized by the ‘578 specification as human MTS1
9 conserved across non-human MTS1 polypeptides. (Paper 41, p. 3, ll. 2-8).
10 Kamb directs the Board’s attention to KX 2016 to support its conservancy
11 argument. KX 2016 is apparently a comparison of human MTS1 with rat
12 and “sus” MTS1.⁶ The three sequences differ in length and in the type of
13 amino acids along the length of the aligned sequences. For example, at
14 aligned position 9, the human sequence contains threonine (T), the rat
15 sequence contains arginine (R) and the sus sequence contains serine (S).
16 Given Kamb’s own statements and evidence, we conclude that one of
17 ordinary skill in the art would understand that mammalian MTS1
18 polypeptides vary in length and can vary in amino acids at a particular
19 aligned position.

20 Kamb cites a single passage from its specification to support its claim
21 construction. (*Id.* at p. 3, ll. 18-19 citing KX 2008 at col. 16, ll. 60-64).
22 Claim construction however, requires that claim terms be read in light of the
23 entire specification not a portion of the specification. Thus, Kamb’s limited

⁶ Kamb fails to direct our attention to a definition for its “Sus_CAC87046” polypeptide described in KX 2016. Dr. Krainer however, identifies KX 2016 as including a pig MTS1 sequence. (BX 1037, ¶ 12). For purposes of this opinion we assume that Kamb’s sus sequence is a mammalian MTS1 polypeptide, most likely pig.

1 citation is simply not enough to demonstrate the correctness of its claim
2 construction. In particular, Kamb should have come forward and sorted out
3 its specification's mishmash of statements regarding polypeptides, MTS1
4 and p16. For example, Kamb's specification states:

5 The term "polypeptide" refers to a polymer of amino acids and
6 its equivalent and does not refer to a specific length of the
7 product; thus, peptides, oligopeptides and proteins are included
8 within the definition of a polypeptide. This term also does not
9 refer to, or exclude modifications of the polypeptide, for
10 example, glycosylations, acetylations, phosphorylations, and
11 the like. *Included within the definition are, for example,*
12 *polypeptides containing one or more analogs of an amino acid*
13 *(including, for example, unnatural amino acids, etc.),*
14 *polypeptides with substituted linkages as well as other*
15 *modifications known in the art, both naturally [sic] and non-*
16 *naturally occurring. Ordinarily, such polypeptides will be at*
17 *least about 50% homologous to the native MTS sequence,*
18 *preferably in excess of about 90%, and more preferably at*
19 *least about 95% homologous.* Also included are proteins
20 encoded by DNA which hybridize under high or low stringency
21 conditions, to MTS-encoding nucleic acids and closely related
22 polypeptides or proteins retrieved by antisera to the MTS
23 protein(s). (KX 2008 at col. 18, ll. 28-50, emphasis added).

24
25 These terms, when applied to a nucleic acid refer to a nucleic
26 acid which encodes a MTS polypeptide (including p16),
27 fragment, homolog or variant, including, e.g., protein fusions or
28 deletions. (Kamb '578 Specification, KX 2008, col. 16, ll. 53-
29 56).

30
31 The term P16 is used interchangeably with MTS1 and MTS1
32 E1 β and is used to mean both MTS1 which encodes a p16 and
33 MTS1E1 β which encodes a p10. MTS1 and MTS1E1 β are two
34 forms of one gene, the two forms being dependent upon which
35 of two promoters is used for transcription. MTS2 is a separate
36 portion of the MTS region and it encodes a p15. (*Id.* at col. 17,
37 ll. 2-8).

1 Recently several negative regulators have also been identified
2 including **p16**, p15, p18, p20, p21 and p27 [citation to Serano
3 et. al. 1993 as well as other publications]. These negative
4 regulators appear to act by inhibiting the kinase activity of the
5 CDKs. Some of the cell cycle regulators are involved in human
6 cancers (for review, see Hunter and Pines, 1994). p20 inhibits
7 Cdk2 and possibly other Cdks while **p16 (also called MTS1,**
8 **CDKN2, or INK4a)** inhibits Cdk4 but apparently does not
9 inhibit Cdk2 in an in vitro assay (Serano et al., 1993). (*Id.* at
10 col. 44, ll. 16-19, emphasis added).

11
12 Additionally, Kamb's claim construction is inconsistent with its
13 noninvolved claims. Specifically, Kamb claims 1 and 4 are not
14 involved in this interference. Kamb claim 1 refers to a "human MTS1
15 polypeptide" that corresponds "to that shown in SEQ ID NO:2."
16 Kamb claim 4 is directed to an isolated amino acid sequence set forth
17 in SEQ ID NO:2. (Kamb Clean Set of Claims, Paper 7). Kamb
18 claims 1 and 4 demonstrate that Kamb was fully capable of limiting
19 its polypeptide to SEQ ID NO:2.

20 We do not credit Kamb's proposed claim construction. We
21 provide claims with their broadest reasonable interpretation in light of
22 the specification as it would be understood by a person of ordinary
23 skill in the art. Kamb's specification states that the term polypeptide
24 refers to a polymer of amino acids and its "equivalent" and goes on to
25 state that one or more analogs of an amino acid are included.
26 (KX 2008, col. 18, ll. 32-43). Kamb's specification also states that
27 polypeptides having at least about 50% homology to the native MTS
28 sequence are included. (*Id.* at col. 18, ll. 43-46). Further, Kamb's
29 own evidence (KX 2016) demonstrates that mammalian MTS1
30 polypeptides vary in length and in the types of amino acids appearing

1 at a particular position. Also, Kamb claims 1 and 4 demonstrate that
2 Kamb could have, but did not, draft claim language expressly limiting
3 its claims to polypeptides shown in SEQ ID. NO: 2.

4

9 Beach contends that its p16 polypeptide is an MTS1 polypeptide.
10 (Beach Motion 2, Paper 42, p. 4, heading line 18). Beach states that Kamb's
11 patent specification uses the terms p16 and MTS1 interchangeably and
12 equates Kamb's MTS1 to Serano's p16. (*Id.* at p. 4, line 24 to p. 5, line 9).
13 Beach also contends that Kamb has defined its MTS1 polypeptide as
14 including variants or fragments with only 50% homology to the native MTS
15 sequence and that Beach's p16 is 94% identical to Kamb's SEQ ID NO: 2.
16 (*Id.* at p. 5, ll. 10-25).

17 Kamb’s specification fails to clearly state the alleged
18 interchangeability of MTS1 polypeptide and p16 polypeptide. Kamb’s
19 specification provides a confusing mishmash of statements regarding the
20 relationship between MTS1 and p16. Part of this confusion stems from
21 Kamb’s use of the terms p16 and MTS1 without identifying whether they
22 are referring to the gene or the polypeptide forms. For example, Kamb
23 states that p16 is also called MTS1 but does not state whether Kamb is
24 speaking of the gene or peptide. (KX 2008, col. 44, lines 16-19). Also,
25 Kamb’s specification states that the MTS1 protein is proposed to participate
26 in a regulatory pathway and cites the Serano 1993 article. (KX 2008, col.
27 55, ll. 3-6). The cited paragraph however concludes with a statement
28 discussing MTS1 and MTS2 expression, which are properties of the genes

1 and not the polypeptides. (*Id.* at col. 53, ll. 17-18 “Thus, in contrast to
2 MTS1, MTS2 expression may be independent of Rb.”). We conclude that
3 Beach has failed to provide sufficient evidence to demonstrate that Kamb’s
4 specification intended to use the terms “MTS1 polypeptide” and “p16
5 polypeptide” interchangeably.

6 Beach contends that Kamb’s MTS1 polypeptide includes variants and
7 fragments, and as such, encompasses Beach’s p16 polypeptide. Beach relies
8 upon the testimony of Dr. Krainer to support its contention. (Paper 42, p. 5,
9 ll. 10-25). Dr. Krainer has been the scientific head of the Cold Spring
10 Harbor Laboratory Cancer Center Antibody Shared Resource facility since
11 1991. Dr. Krainer has authored of over one hundred peer-reviewed
12 publications in the area of molecular biology since 1981 and has served on
13 various molecular biology editorial boards. (BX 1004, ¶¶ 4-6). We find that
14 Dr. Krainer is qualified to testify as to knowledge and understanding
15 possessed by one of ordinary skill in the art, the art being antibodies that are
16 immunoreactive to a protein.

17 Dr. Krainer states that Kamb’s mammalian MTS1 polypeptide
18 encompasses the human p16 described in Serano 1993 and in the Beach ‘915
19 application and in Beach’s ‘169 patent. (*Id.* at ¶ 20). Dr. Krainer’s
20 reasoning is as follows. Beach ‘915 and Serano 1993 describes the same
21 sequence for human p16 polypeptide. (*Id.* at ¶ 11). The human p16
22 polypeptide is identical to residues 9-156 of Kamb’s SEQ ID NO:2 (human
23 MTS1), except that Kamb has glycine at position 35 whereas Beach and
24 Serano have valine at corresponding position 27. (*Id.* at ¶ 13). Dr. Krainer
25 testifies that Kamb’s specification fails to specify a particular method for
26 calculating homology or identity between two sequences, but that such
27 calculations were known in the art. (*Id.* at ¶ 18). Dr. Krainer testifies that

1 using the typical method of calculation, the percent identity between Kamb's
2 MTS1 polypeptide (SEQ ID NO: 2) and Beach and Serano's p16 is 94%.⁷
3 (*Id.* at ¶ 19). Dr. Krainer testifies that percent homology always provides a
4 value equal to or greater than percent identity between two sequences. (*Id.*
5 at ¶ 18). Dr. Krainer concludes that the percent homology between Kamb's
6 MTS1 polypeptide and Beach and Serano's p16 is 94% or greater. (*Id.* at ¶
7 19).

8 We credit Dr. Krainer’s testimony that Beach and Serano’s human
9 p16 polypeptide falls within the scope of Kamb’s claimed mammalian
10 MTS1 polypeptide. Dr. Krainer’s testimony is consistent with the evidence
11 of record, and in particular, consistent with the teachings of Kamb’s
12 specification. Kamb’s specification describes its polypeptides as
13 encompassing variants and fragments including polypeptides that are at least
14 about 50% homologous to the native MTS sequence, preferably in excess of
15 about 90% and more preferably in excess of about 95%. (KX 2008, col. 18,
16 ll. 28-50). Beach and Serano’s polypeptide are at least 94% homologous
17 with Kamb’s MTS1 polypeptide and thus fall within Kamb’s preferred range
18 of polypeptide homology.

19

b. Immunoreactive Antibodies

21 Kamb's Opposition 2 contends that Beach's antibodies are cross-
22 reactive with p15 and p15.5 and therefore Beach lacks an enabling
23 disclosure of a "specifically immunoreactive" antibody. (Paper 42, p. 4, ll.
24 7-19). Beach counters arguing that exclusion of all cross-reactivity is an

⁷ Dr. Krainer arrived at the 94% value by dividing the number of identical amino acids in the sequence (147) by the total number of amino acids in Kamb's SEQ ID NO:2. (157). (*Id.* at ¶ 19).

1 unreasonably narrow claim construction. (Paper 50, p. 4, ll. 8-16).

2 Kamb appears to take the position that Beach cannot anticipate
3 Kamb's claims unless Beach enables a "specifically" immunoreactive
4 antibody. Kamb claims 2 and 3 require antibodies that are immunoreactive
5 with a mammalian MTS1 polypeptide but not immunoreactive with other
6 mammalian polypeptides.

7 Kamb's claims do not require the antibody to be "specifically"
8 immunoreactive. Kamb fails to state how the term "specifically"
9 immunoreactive" antibodies compares with the claimed "immunoreactive"
10 antibodies. To the extent there is a difference between the terms, Kamb does
11 not direct our attention to credible evidence of record to demonstrate that its
12 immunoreactive antibodies must be specifically immunoreactive.⁸
13 Accordingly, giving Kamb's claims their broadest reasonable interpretation
14 we will not read the limitation "specifically" into the claims.

15 Kamb implicitly construes the term "immunoreactive" to exclude
16 weak cross-reactivity. For example, Beach's involved '869 application
17 describes weak cross-reactivity between p16 antiserum and p15 and p15.5.
18 (KX 2005, p. 55, '932 publication para. 189). Kamb states that Beach fails
19 to provide a description about methods of making antibodies that can
20 distinguish between p16 protein and p15 or p15.5. (Paper 42, p. 4, ll. 12-
21 14). Kamb concludes that Beach cannot anticipate Kamb's claims without
22 such a disclosure. (*Id.* at p. 4, ll. 14-19).

23 Beach argues that the term immunoreactive allows for recognition of

⁸Kamb Opposition 2 cites only three exhibits, Beach's 915 application (KX 2002), Beach's '392 publication and Kamb's involved '578 patent (KX 2008). Kamb Opposition 2 does not explain how these references compel a conclusion that Kamb's immunoreactive antibodies must be "specifically" immunoreactive.

1 some amount of crossreactivity. (Paper 50, p. 4, ll. 17-23). Beach, relying
2 on its expert Dr. Krainer, contends that one of ordinary skill would
3 understand that it is scientifically difficult to obtain an antibody having
4 absolutely no cross-reactivity because an antibody is likely to recognize a
5 small region on one of the millions of other mammalian polypeptides that
6 overlap a small region on the antibody-binding site of MTS1. (*Id.* at p. 4, ll.
7 19-23 citing Dr. Krainer Dec. KX 1037, ¶ 4).

8 The starting point for claim interpretation is from the vantage point of
9 the person of ordinary skill in the art. *Phillips v. AWH Corp.* 415 F.3d 1303,
10 1313 (Fed. Cir. 2005)(en banc). As discussed above, Dr. Krainer is qualified
11 to testify as to the understanding of a person of ordinary skill in this art. Dr.
12 Krainer testifies that Beach describes antibodies that are immunoreactive
13 with p16, as well as antibodies that are specifically immunoreactive with
14 p16 as those terms are used in Kamb's claims. (KX 1005, ¶¶ 23, 45). Kamb
15 has failed to direct our attention to credible evidence of record that
16 contradicts Dr. Krainer's interpretation of Kamb's claims and the prior art.
17 Based upon the record presented, we credit the testimony of Dr. Krainer and
18 conclude that Kamb's claimed "immunoreactive" antibodies encompass
19 antibodies that react with mammalian MTS1 polypeptides but have weak
20 cross-reactivity with non mammalian MTS1 polypeptides.

21
22 2. Beach's '169 Patent and '915 Application Provide a
23 Described and Enabled Embodiment within the Scope of
24 Kamb Claims 2 and 3
25

26 Beach contends that its '169 patent and '915 application describe and
27 enable a monoclonal antibody that is immunoreactive with a mammalian
28 MTS1 polypeptide, p16, but not to other mammalian MTS1 polypeptides.

1 Beach's expert, Dr. Krainer, testifies that both the Beach '169 patent and the
2 '915 application explicitly state that an aspect of the invention is the
3 formation of an antibody that is specifically reactive with p16 protein.
4 (KX 1004, ¶ 44, citing Beach '169, KX 1007, col. 20, lines 46-51 and
5 Beach '915, KX 1008, p. 27, ll. 19-20). Dr. Krainer cites specific teachings
6 in the '169 patent and '915 application and concludes that they describe the
7 experimental preparation and use of antibodies that are immunoreactive with
8 a p16 polypeptide. (KX 1004, ¶¶ 41-45). Dr. Krainer testifies that both the
9 '169 patent and '915 application describe using known techniques for the
10 generation of monoclonal antibodies. (*Id.* at ¶ 46). Dr. Krainer also testifies
11 that even an ordinary graduate student studying molecular biology could
12 have, without undue experimentation, followed the disclosures of the '169
13 patent and '915 application and produced antibodies specifically
14 immunoreactive with a p16 polypeptide. (*Id.* at ¶ 47).

15 Kamb presents two arguments in opposition. Specifically, Kamb
16 argues that Beach's '169 patent and '915 application do not describe a
17 MTS1 polypeptide and that they do not enable antibodies that are
18 "specifically immunoreactive" with p16 or MTS1 polypeptide. (Paper 42,
19 bolded headings A and B). For the reasons presented below, we do not
20 credit these arguments.

21 Kamb states that its mammalian MTS1 polypeptide is "clearly defined
22 to be SEQ ID NO: 2" and that this definition "unequivocally lacks fragments
23 and variants." (Paper 42, p. 3, ll. 18-20). As discussed in detail above,
24 Kamb has failed to provide credible and sufficient evidence to support its
25 position. Indeed, Kamb's own arguments and evidence demonstrate that
26 mammalian MTS1 polypeptides are not limited to Kamb's SEQ ID NO: 2
27 and that the sequences vary in length and can possess different amino acids

1 at a particular position in the sequence. (KX 2016). Based upon the
2 evidence presented, we hold that Kamb’s “mammalian MTS1 polypeptide”
3 does not exclude fragments and variants, such as human p16.

4 Kamb alleges that Beach’s ‘169 patent and ‘915 application do not
5 enable antibodies that are specifically immunoreactive with p16 or MTS1
6 polypeptide. (Paper 42, p. 4, bolded heading). Kamb bears the burden of
7 establishing the nonenablement of the prior art asserted against its claims.

8 *C.f., Amgen, Inc. v. Hoechst Marion Roussel, Inc.*, 314 F.3d 1313, 1355
9 (Fed. Cir. 2003) (“We hold that an accused infringer should be similarly
10 entitled to have the district court presume the enablement of unclaimed (and
11 claimed) material in a prior art patent defendant asserts against a plaintiff.”).

12 Kamb’s allegations regarding a lack of enablement are based upon its
13 counsel’s interpretation of Beach’s specifications. Yet, enablement must be
14 placed in the context of how one of ordinary skill in the art would have
15 interpreted the disclosure. Specifically, the scope of enablement is that
16 which is disclosed plus the scope of what would be known to one of
17 ordinary skill in the art without undue experimentation. Beach’s expert, Dr.
18 Krainer, testifies that even an ordinary graduate student could have produced
19 antibodies specifically immunoreactive with a p16 polypeptide given the
20 disclosure of Beach’s ‘169 patent and ‘915 application. (*Id.* at ¶ 47).

21 Weighing Dr. Krainer’s testimony against Kamb’s attorney argument we
22 credit Dr. Krainer testimony and not the arguments of Kamb’s counsel.

23 We **grant** Beach Substantive Motion 2 and hold that Kamb’s involved
24 claims, claims 2 and 3, are unpatentable as anticipated under 35 U.S.C. §
25 102(e).

1 D. Priority of Invention is Awarded Against Junior Party Kamb
2 Kamb's earliest accorded priority date is March 18, 1994. (Notice
3 Declaring Interference, Paper 1, p. 5). Beach's earliest accorded priority
4 date is November 18, 1993, based upon its '915 application.

5 Kamb claim 2, which forms a part of Count 1, has been held
6 unpatentable as anticipated under 35 U.S.C. §102(e) by Beach's '169 patent.
7 The '169 patent is available as prior art to Kamb under §102(e) based upon
8 its claimed 35 U.S.C. § 120 benefit of Beach's earlier filed '915 application,
9 filed November 18, 1993. Kamb did not attempt to antedate Beach's '169
10 patent § 102(e) effective filing date of November 18, 1993. Furthermore,
11 junior party Kamb did not file a priority statement that alleges a date of
12 conception that antedates Beach's accorded benefit date of November 18,
13 1993.

14 The remaining claims of Kamb '578, claims 1 and 4, are directed to
15 protein compositions comprising a polypeptide having Kamb's SEQ ID NO:
16 2 amino acid sequence. These claims were designated as not corresponding
17 to Count 1. Accordingly, Kamb no longer has any patentable claims in the
18 interference that correspond to Count 1.

19 Based on the facts of this case, the Board enters judgment on priority
20 against junior party Kamb. 37 C.F.R. § 41.204. Judgment will be entered
21 concurrent with this decision in a separate paper against junior party Kamb.
22 Since no there is priority phase in this interference, there is no occasion to
23 file motions associated with that phase.

24
25 E. Beach Substantive Motions 1, 3 and 4 for Judgment are
26 Dismissed as Moot
27
28 Beach Motions 1 and 4 request judgment against Kamb claims 2 and 3

1 based upon prior art. (Papers 26 and 29). Kamb opposes. (Papers 40 and
2 41). Kamb claims 2 and 3 have already been held unpatentable over prior
3 art and lack of priority of invention. Accordingly, we dismiss Beach
4 Motions 1 and 4 as *moot*.

5 Beach Motion 3 requests that Kamb claims 2 and 3 be held
6 unpatentable for lack of utility under 35 U.S.C § 101. (Paper 28). Kamb
7 opposes. (Paper 43). As Kamb claims 2 and 3 have already been held
8 unpatentable to Kamb, we dismiss Beach Motion 3 as *moot*.

9

10 IV. ORDER

11 Based upon the evidence identified in the record, it is:

12 **Ordered** that Beach Substantive Motion 2, is *granted* with respect to
13 its request that all of Kamb's involved claims, claims 2 and 3, be held
14 unpatentable over prior art.

15 **Further Ordered** that Beach Substantive Motion 1, which requests
16 that Kamb claims 2 and 3 be held unpatentable over prior art, is dismissed as
17 *moot*.

18 **Further Ordered** that Beach Substantive Motion 4, which requests
19 that Kamb claims 2 and 3 be held unpatentable over prior art, is dismissed as
20 *moot*.

21 **Further Ordered** that Beach Substantive Motion 3, which requests
22 that Kamb claims 2 and 3 be held unpatentable for lack of utility under 35
23 U.S.C §101, is dismissed as *moot*.

24 **Further Ordered** that Beach Responsive Contingent Motion, which
25 requests that the count be redefined, is dismissed as *moot*.

26 **Further Ordered** that Kamb Substantive Motion 1, which requests
27 that Beach's involved claims be held unpatentable for lack of written

1 description and/or enablement under 35 U.S.C. 112, first paragraph is
2 ***denied.***

3

4

5

6 Michael P. Tierney/)
7 MICHAEL P. TIERNEY)
8 Administrative Patent Judge)

9

10

11 James T. Moore/)
12 JAMES T. MOORE)
13 Administrative Patent Judge)

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